CHAPTER 84

Sports Physiology

There are few stresses to which the body is exposed that approach the extreme stresses of heavy exercise. In fact, if some of the extremes of exercise were continued for even moderately prolonged periods, they might be lethal. Therefore, sports physiology is mainly a discussion of the ultimate limits to which several of the bodily mechanisms can be stressed. To give one simple example: In a person who has extremely high fever approaching the level of lethality, the body metabolism increases to about 100 percent above normal. By comparison, the metabolism of the body during a marathon race may increase to 2000 percent above normal.

Female and Male Athletes

Most of the quantitative data that are given in this chapter are for the young male athlete, not because it is desirable to know only these values but because it is only in male athletes that relatively complete measurements have been made. However, for those measurements that have been made in the female athlete, similar basic physiologic principles apply, except for quantitative differences caused by differences in body size, body composition, and the presence or absence of the male sex hormone testosterone.

In general, most quantitative values for women—such as muscle strength, pulmonary ventilation, and cardiac output, all of which are related mainly to the muscle mass—vary between two thirds and three quarters of the values recorded in men. When measured in terms of strength per square centimeter of cross-sectional area, the female muscle can achieve almost exactly the same maximal force of contraction as that of the male—between 3 and 4 kg/cm^2. Therefore, most of the difference in total muscle performance lies in the extra percentage of the male body that is muscle, caused by endocrine differences that we discuss later.

The performance capabilities of the female versus male athlete are illustrated by the relative running speeds for a marathon race. In a comparison, the top female performer had a running speed that was 11 percent less than that of the top male performer. For other events, however, women have at times held records faster than men—for instance, for the two-way swim across the English Channel, where the availability of extra fat seems to be an advantage for heat insulation, buoyancy, and extra long-term energy.

Testosterone secreted by the male testes has a powerful *anabolic effect* in causing greatly increased deposition of protein everywhere in the body, but especially in the muscles. In fact, even a male who participates in very little sports activity but who nevertheless has a normal level of testosterone will have muscles that grow about 40 percent larger than those of a comparable female without the testosterone.

The female sex hormone *estrogen* probably also accounts for some of the difference between female and male performance, although not nearly so much as testosterone. Estrogen increases the deposition of fat in the female, especially in the breasts, hips, and subcutaneous tissue. At least partly for this reason, the average nonathletic female has about 27 percent body fat composition, in contrast to the nonathletic male, who has about 15 percent. This is a detriment to the highest levels of athletic performance in those events in which performance depends on speed or on ratio of total body muscle strength to body weight.

Muscles in Exercise

Strength, Power, and Endurance of Muscles

The final common determinant of success in athletic events is what the muscles can do for you—what strength they can give when it is needed, what power they can achieve in the performance of work, and how long they can continue their activity.

The strength of a muscle is determined mainly by its size, with a *maximal contractile force between 3 and 4 kg/cm*^{2} of muscle cross-sectional area. Thus, a man who is well supplied with testosterone or who has enlarged his muscles through an exercise training program will have correspondingly increased muscle strength.

To give an example of muscle strength, a world-class weight lifter might have a quadriceps muscle with a cross-sectional area as great as 150 square centimeters. This would translate into a maximal contractile strength of 525 kilograms (or 1155 pounds), with all this force applied to the patellar tendon. Therefore, one can readily understand how it is possible for this tendon at times to be ruptured or actually to be avulsed from its insertion into the tibia below the knee. Also, when such forces occur in tendons that span a joint, similar forces are applied to the surfaces of the joint or sometimes to ligaments spanning the joints, thus accounting for such happenings as displaced cartilages, compression fractures about the joint, and torn ligaments.

The *holding strength* of muscles is about 40 percent greater than the contractile strength. That is, if a muscle is already contracted and a force then attempts to stretch out the muscle, as occurs when landing after a jump, this requires about 40 percent more force than can be achieved by a shortening contraction. Therefore, the force of 525 kilograms calculated above for the patellar tendon during muscle contraction becomes 735 kilograms (1617 pounds) during holding contractions. This further compounds the problems of the tendons, joints, and ligaments. It can also lead to internal tearing in the muscle itself. In fact, forceful stretching of a maximally contracted muscle is one of the surest ways to create the highest degree of muscle soreness.

Mechanical work performed by a muscle is the amount of force applied by the muscle multiplied by the distance over which the force is applied. The *power* of muscle contraction is different from muscle strength because power is a measure of the total amount of work that the muscle performs in a unit period of time. Power is therefore determined not only by the strength of muscle contraction but also by its *distance of contraction* and the *number of times that it contracts each minute.* Muscle power is generally measured in *kilogram meters (kg-m) per minute.* That is, a muscle that can lift 1 kilogram weight to a height of 1 meter or that can move some object laterally against a force of 1 kilogram for a distance of 1 meter in 1 minute is said to have a power of 1 kg-m/min. The maximal power achievable by all the muscles in the body of a highly trained athlete with all the muscles working together is approximately the following:

	kg-m/min
First 8 to 10 seconds	7000
Next 1 minute	4000
Next 30 minutes	1700

Thus, it is clear that a person has the capability of extreme power surges for short periods of time, such as during a 100-meter dash that is completed entirely within 10 seconds, whereas for long-term endurance events, the power output of the muscles is only one fourth as great as during the initial power surge.

This does not mean that one's athletic performance is four times as great during the initial power surge as it is for the next 30 minutes, because the *efficiency* for translation of muscle power output into athletic performance is often much less during rapid activity than during less rapid but sustained activity. Thus, the velocity of the 100-meter dash is only 1.75 times as great as the velocity of a 30-minute race, despite the fourfold difference in short-term versus long-term muscle power capability.

Another measure of muscle performance is *endurance.* This, to a great extent, depends on the nutritive support for the muscle—more than anything else on the amount of glycogen that has been stored in the muscle before the period of exercise. A person on a high-carbohydrate diet stores far more glycogen in muscles than a person on either a mixed diet or a high-fat diet. Therefore, endurance is greatly enhanced by a high-carbohydrate diet. When athletes run at speeds typical for the marathon race, their endurance (as measured by the time that they can sustain the race until complete exhaustion) is approximately the following:

	Minutes
High-carbohydrate diet	240
Mixed diet	120
High-fat diet	85

The corresponding amounts of glycogen stored in the muscle before the race started explain these differences. The amounts stored are approximately the following:

	g/kg Muscle
High-carbohydrate diet	40
Mixed diet	20
High-fat diet	6

Muscle Metabolic Systems in Exercise

The same basic metabolic systems are present in muscle as in other parts of the body; these are discussed in detail in Chapters 67 through 73. However, special quantitative measures of the activities of three metabolic systems are exceedingly important in understanding the limits of physical activity. These systems are (1) *the phosphocreatine-creatine system,* (2) *the glycogen-lactic acid system,* and (3) *the aerobic system.*

Adenosine Triphosphate. The source of energy actually used to cause muscle contraction is adenosine triphosphate (ATP), which has the following basic formula:

$$\text{Adenosine-PO}_3 \sim \text{PO}_3 \sim \text{PO}_3^-$$

The bonds attaching the last two phosphate radicals to the molecule, designated by the symbol ~, are *high-energy phosphate bonds.* Each of these bonds stores 7300 calories of energy per mole of ATP under standard conditions (and even slightly more than this under the physical conditions in the body, which is discussed in detail in Chapter 67). Therefore, when one phosphate radical is removed, more than 7300 calories of energy are released to energize the muscle contractile process. Then, when the second phosphate radical is removed, still another 7300 calories become available. Removal of the first phosphate converts the ATP into *adenosine diphosphate* (ADP), and removal of the second converts this ADP into *adenosine monophosphate* (AMP).

The amount of ATP present in the muscles, even in a well-trained athlete, is sufficient to sustain maximal muscle power for only about 3 seconds, maybe enough for one half of a 50-meter dash. Therefore, except for a few seconds at a time, it is essential that new ATP be formed continuously, even during the performance of short athletic events. Figure 84-1 shows the overall metabolic system, demonstrating the breakdown of ATP first to ADP and then to AMP, with the release of energy to the muscles for contraction. The left-hand side of the figure shows the three metabolic systems that provide a continuous supply of ATP in the muscle fibers.

Phosphocreatine-Creatine System

Phosphocreatine (also called *creatine phosphate*) is another chemical compound that has a high-energy phosphate bond, with the following formula:

$$\text{Creatine} \sim \text{PO}_3^-$$

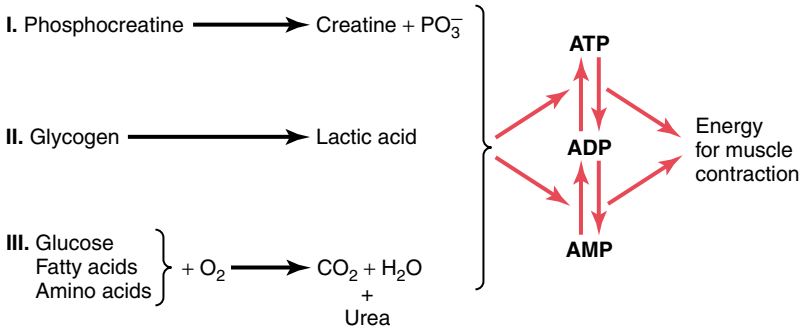

Figure 84-1 Important metabolic systems that supply energy for muscle contraction.

This can decompose to *creatine* and *phosphate ion,* as shown in Figure 84-1, and in doing so releases large amounts of energy. In fact, the high-energy phosphate bond of phosphocreatine has more energy than the bond of ATP, 10,300 calories per mole compared with 7300 for the ATP bond. Therefore, phosphocreatine can easily provide enough energy to reconstitute the high-energy bond of ATP. Furthermore, most muscle cells have two to four times as much phosphocreatine as ATP.

A special characteristic of energy transfer from phosphocreatine to ATP is that it occurs within a small fraction of a second. Therefore, all the energy stored in the muscle phosphocreatine is almost instantaneously available for muscle contraction, just as is the energy stored in ATP.

The combined amounts of cell ATP and cell phosphocreatine are called the *phosphagen energy system.* These together can provide maximal muscle power for 8 to 10 seconds, almost enough for the 100-meter run. *Thus, the energy from the phosphagen system is used for maximal short bursts of muscle power.*

Glycogen-Lactic Acid System. The stored glycogen in muscle can be split into glucose and the glucose then used for energy. The initial stage of this process, called *glycolysis,* occurs without use of oxygen and, therefore, is said to be *anaerobic metabolism* (see Chapter 67). During glycolysis, each glucose molecule is split into two *pyruvic acid molecules,* and energy is released to form four ATP molecules for each original glucose molecule, as explained in Chapter 67. Ordinarily, the pyruvic acid then enters the mitochondria of the muscle cells and reacts with oxygen to form still many more ATP molecules. However, when there is insufficient oxygen for this second stage (the oxidative stage) of glucose metabolism to occur, most of the pyruvic acid then is converted into *lactic acid,* which diffuses out of the muscle cells into the interstitial fluid and blood. Therefore, much of the muscle glycogen is transformed to lactic acid, but in doing so, considerable amounts of ATP are formed entirely without the consumption of oxygen.

Another characteristic of the glycogen-lactic acid system is that it can form ATP molecules about 2.5 times as rapidly as can the oxidative mechanism of the mitochondria. Therefore, when large amounts of ATP are required for short to moderate periods of muscle contraction, this anaerobic glycolysis mechanism can be used as a rapid source of energy. It is, however, only about one half as rapid as the phosphagen system. Under optimal conditions, the glycogen-lactic acid system can provide 1.3 to 1.6 minutes of maximal muscle activity in addition to the 8 to 10 seconds provided by the phosphagen system, although at somewhat reduced muscle power.

Aerobic System. The aerobic system is the oxidation of foodstuffs in the mitochondria to provide energy. That is, as shown to the left in Figure 84-1, glucose, fatty acids, and amino acids from the foodstuffs—after some intermediate processing—combine with oxygen to release tremendous amounts of energy that are used to convert AMP and ADP into ATP, as discussed in Chapter 67.

In comparing this aerobic mechanism of energy supply with the glycogen-lactic acid system and the phosphagen system, the relative *maximal rates of power generation* in terms of moles of ATP generation per minute are the following:

	Moles of ATP/min
Phosphagen system	4
Glycogen-lactic acid system	2.5
Aerobic system	1

When comparing the same systems for endurance, the relative values are the following:

	Time
Phosphagen system	8-10 seconds
Glycogen-lactic acid system	1.3-1.6 minutes
Aerobic system	Unlimited time (as long as nutrients last)

Thus, one can readily see that the phosphagen system is the one used by the muscle for power surges of a few seconds, and the aerobic system is required for prolonged athletic activity. In between is the glycogen-lactic acid system, which is especially important for giving extra power during such intermediate races as the 200- to 800-meter runs.

What Types of Sports Use Which Energy Systems? By considering the vigor of a sports activity and its duration, one can estimate closely which of the energy systems is used for each activity. Various approximations are presented in Table 84-1.

Recovery of the Muscle Metabolic Systems After Exercise. In the same way that the energy from phosphocreatine can be used to reconstitute ATP, energy from the glycogen-lactic acid system can be used to reconstitute both phosphocreatine and ATP. And then energy from the oxidative metabolism of the aerobic system can be used to reconstitute all the other systems—the ATP, the phosphocreatine, and the glycogen-lactic acid system.

Table 84-1 Energy Systems Used in Various Sports

Phosphagen System, Almost Entirely
100-meter dash
Jumping
Weight lifting
Diving
Football dashes
Baseball triple
Phosphagen and Glycogen-Lactic Acid Systems
200-meter dash
Basketball
Ice hockey dashes
Glycogen-Lactic Acid System, Mainly
400-meter dash
100-meter swim
Tennis
Soccer
Glycogen-Lactic Acid and Aerobic Systems
800-meter dash
200-meter swim
1500-meter skating
Boxing
2000-meter rowing
1500-meter run
1-mile run
400-meter swim
Aerobic System
10,000-meter skating
Cross-country skiing
Marathon run (26.2 miles, 42.2km)
Jogging

Reconstitution of the lactic acid system means mainly the removal of the excess lactic acid that has accumulated in all the fluids of the body. This is especially important because *lactic acid causes extreme fatigue.* When adequate amounts of energy are available from oxidative metabolism, removal of lactic acid is achieved in two ways: (1) A small portion of it is converted back into pyruvic acid and then metabolized oxidatively by all the body tissues. (2) The remaining lactic acid is reconverted into glucose mainly in the liver, and the glucose in turn is used to replenish the glycogen stores of the muscles.

Recovery of the Aerobic System After Exercise. Even during the early stages of heavy exercise, a portion of one's aerobic energy capability is depleted. This results from two effects: (1) the so-called *oxygen debt* and (2) *depletion of the glycogen stores* of the muscles.

Oxygen Debt. The body normally contains about 2 liters of stored oxygen that can be used for aerobic metabolism even without breathing any new oxygen. This stored oxygen consists of the following: (1) 0.5 liter in the air of the lungs, (2) 0.25 liter dissolved in the body fluids, (3) 1 liter combined with the hemoglobin of the blood, and (4) 0.3 liter stored in the muscle fibers themselves, combined mainly with myoglobin, an oxygen-binding chemical similar to hemoglobin.

In heavy exercise, almost all this stored oxygen is used within a minute or so for aerobic metabolism. Then, after the exercise is over, this stored oxygen must be replenished by breathing extra amounts of oxygen over and above the normal requirements. In addition, about 9 liters more oxygen must be consumed to provide for reconstituting both the phosphagen system and the lactic acid system. All this extra oxygen that must be "repaid," about 11.5 liters, is called the oxygen debt.

Figure 84-2 shows this principle of oxygen debt. During the first 4 minutes of the figure, the person exercises heavily, and the rate of oxygen uptake increases more than 15-fold. Then, even after the exercise is over, the oxygen uptake still remains above normal, at first very high while the body is reconstituting the phosphagen system and repaying the stored oxygen portion of the oxygen debt, and then for another 40 minutes at a lower level while the lactic acid is removed. The early portion of the oxygen debt is called the *alactacid oxygen debt* and amounts to about 3.5 liters. The latter portion is called the *lactic acid oxygen debt* and amounts to about 8 liters.

Recovery of Muscle Glycogen. Recovery from exhaustive muscle glycogen depletion is not a simple matter. This often requires days, rather than the seconds, minutes, or hours required for recovery of the phosphagen and lactic acid metabolic systems. Figure 84-3 shows this recovery process under three conditions: first, in people on a high-carbohydrate diet; second, in people on a high-fat, high-protein diet; and third, in people with no food. Note that on a high-carbohydrate diet, full recovery occurs in about 2 days. Conversely, people on a high-fat, high-protein diet or on no food at all show very little recovery even after as long as 5 days. The messages of this comparison are (1) that it is important for an athlete to have a high-carbohydrate diet before a grueling athletic event and (2) not to participate in exhaustive exercise during the 48 hours preceding the event.

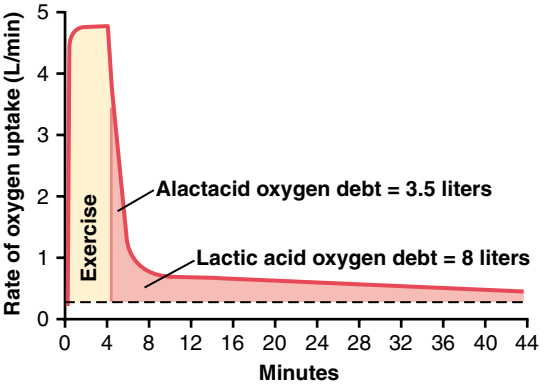

Figure 84-2 Rate of oxygen uptake by the lungs during maximal exercise for 4 minutes and then for about 40 minutes after the exercise is over. This figure demonstrates the principle of *oxygen debt.*

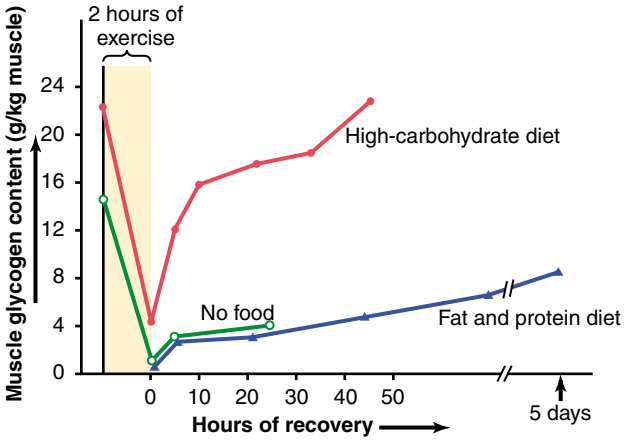

Figure 84-3 Effect of diet on the rate of muscle glycogen replenishment after prolonged exercise. (Redrawn from Fox EL: Sports Physiology. Philadelphia: Saunders College Publishing, 1979.)

Nutrients Used During Muscle Activity

In addition to the large usage of carbohydrates by the muscles during exercise, especially during the early stages of exercise, muscles use large amounts of fat for energy in the form of *fatty acids* and *acetoacetic acid* (see Chapter 68), and they use to a much less extent proteins in the form of *amino acids*. In fact, even under the best conditions, in endurance athletic events that last longer than 4 to 5 hours, the glycogen stores of the muscle become almost totally depleted and are of little further use for energizing muscle contraction. Instead, the muscle now depends on energy from other sources, mainly from fats.

Figure 84-4 shows the approximate relative usage of carbohydrates and fat for energy during prolonged exhaustive exercise under three dietary conditions: high-carbohydrate diet, mixed diet, and high-fat diet. Note that most of the energy is derived from carbohydrates during the first few seconds or minutes of the exercise, but at the time of exhaustion, as much as 60 to 85 percent of the energy is being derived from fats, rather than carbohydrates.

Not all the energy from carbohydrates comes from the stored *muscle* glycogen. In fact, almost as much glycogen is stored in the *liver* as in the muscles, and this can be released into the blood in the form of glucose and then taken up by the muscles as an energy source. In addition, glucose solutions given to an athlete to drink during the course of an athletic event can provide as much as 30 to 40 percent of the energy required during prolonged events such as marathon races.

Therefore, if muscle glycogen and blood glucose are available, they are the energy nutrients of choice for intense muscle activity. Even so, for a long-term endurance event, one can expect fat to supply more than 50 percent of the required energy after about the first 3 to 4 hours.

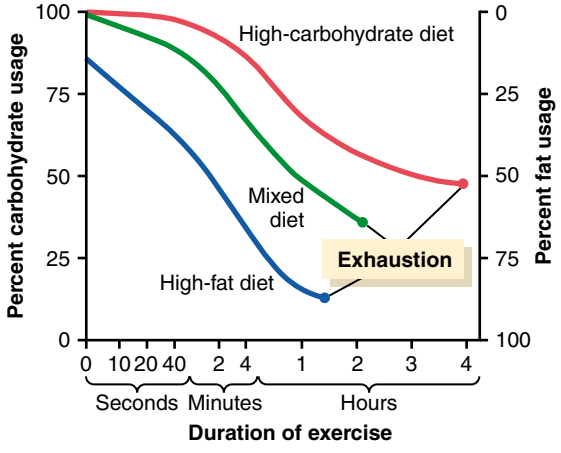

Figure 84-4 Effect of duration of exercise, as well as type of diet on relative percentages of carbohydrate or fat used for energy by muscles. (Based partly on data in Fox EL: Sports Physiology. Philadelphia: Saunders College Publishing, 1979.)

Effect of Athletic Training on Muscles and Muscle Performance

Importance of Maximal Resistance Training. One of the cardinal principles of muscle development during athletic training is the following: Muscles that function under no load, even if they are exercised for hours on end, increase little in strength. At the other extreme, muscles that contract at more than 50 percent maximal force of contraction will develop strength rapidly even if the contractions are performed only a few times each day. Using this principle, experiments on muscle building have shown that *six nearly maximal muscle contractions performed in three sets 3 days a week give approximately optimal increase in muscle strength, without producing chronic muscle fatigue.*

The upper curve in Figure 84-5 shows the approximate percentage increase in strength that can be achieved in a previously untrained young person by this resistive training program, demonstrating that the muscle strength increases about 30 percent during the first 6 to 8 weeks but almost plateaus after that time. Along with this increase in strength is an approximately equal percentage increase in muscle mass, which is called *muscle hypertrophy.*

In old age, many people become so sedentary that their muscles atrophy tremendously. In these instances, muscle training often increases muscle strength more than 100 percent.

Muscle Hypertrophy. The average size of a person's muscles is determined to a great extent by heredity plus the level of testosterone secretion, which, in men, causes considerably larger muscles than in women. With training, however, the muscles can become hypertrophied perhaps an additional 30 to 60 percent. Most of this hypertrophy results from

Figure 84-5 Approximate effect of optimal resistive exercise training on increase in muscle strength over a training period of 10 weeks.

increased diameter of the muscle fibers rather than increased numbers of fibers. However, a very few greatly enlarged muscle fibers are believed to split down the middle along their entire length to form entirely new fibers, thus increasing the number of fibers slightly.

The changes that occur inside the hypertrophied muscle fibers themselves include (1) increased numbers of myofibrils, proportionate to the degree of hypertrophy; (2) up to 120 percent increase in mitochondrial enzymes; (3) as much as 60 to 80 percent increase in the components of the phosphagen metabolic system, including both ATP and phosphocreatine; (4) as much as 50 percent increase in stored glycogen; and (5) as much as 75 to 100 percent increase in stored triglyceride (fat). Because of all these changes, the capabilities of both the anaerobic and the aerobic metabolic systems are increased, increasing especially the maximum oxidation rate and efficiency of the oxidative metabolic system as much as 45 percent.

Fast-Twitch and Slow-Twitch Muscle Fibers. In the human being, all muscles have varying percentages of *fast-twitch* and *slow-twitch muscle fibers.* For instance, the gastrocnemius muscle has a higher preponderance of fast-twitch fibers, which gives it the capability of forceful and rapid contraction of the type used in jumping. In contrast, the soleus muscle has a higher preponderance of slow-twitch muscle fibers and therefore is used to a greater extent for prolonged lower leg muscle activity.

The basic differences between the fast-twitch and the slow-twitch fibers are the following:

1. Fast-twitch fibers are about twice as large in diameter.
2. The enzymes that promote rapid release of energy from the phosphagen and glycogen-lactic acid energy systems are two to three times as active in fast-twitch fibers as in slow-twitch fibers, thus making the maximal power that can be achieved for very short periods of time by fast-twitch fibers about twice as great as that of slow-twitch fibers.
3. Slow-twitch fibers are mainly organized for endurance, especially for generation of aerobic energy. They have far more mitochondria than the fast-twitch fibers. In addition, they contain considerably more myoglobin, a hemoglobin-like protein that combines with oxygen within the muscle fiber; the extra myoglobin increases the rate of diffusion of oxygen throughout the fiber by shuttling oxygen from one molecule of myoglobin to the next. In addition, the enzymes of the aerobic metabolic system are considerably more active in slow-twitch fibers than in fast-twitch fibers.
4. The number of capillaries is greater in the vicinity of slow-twitch fibers than in the vicinity of fast-twitch fibers.

In summary, fast-twitch fibers can deliver extreme amounts of power for a few seconds to a minute or so. Conversely, slow-twitch fibers provide endurance, delivering prolonged strength of contraction over many minutes to hours.

Hereditary Differences Among Athletes for Fast-Twitch Versus Slow-Twitch Muscle Fibers. Some people have considerably more fast-twitch than slow-twitch fibers, and others have more slow-twitch fibers; this could determine to some extent the athletic capabilities of different individuals. Athletic training has not been shown to change the relative proportions of fast-twitch and slow-twitch fibers however much an athlete might want to develop one type of athletic prowess over another. Instead, this seems to be determined almost entirely by genetic inheritance, and this in turn helps determine which area of athletics is most suited to each person: some people appear to be born to be marathoners; others are born to be sprinters and jumpers. For example, the following are recorded percentages of fast-twitch versus slow-twitch fiber in the quadriceps muscles of different types of athletes:

	Fast-Twitch	Slow-Twitch
Marathoners	18	82
Swimmers	26	74
Average male	55	45
Weight lifters	55	45
Sprinters	63	37
Jumpers	63	37

Respiration in Exercise

Although one's respiratory ability is of relatively little concern in the performance of sprint types of athletics, it is critical for maximal performance in endurance athletics.

Oxygen Consumption and Pulmonary Ventilation in Exercise. Normal oxygen consumption for a young man at rest is about 250 ml/min. However, under maximal conditions, this can be increased to approximately the following average levels:

	ml/min
Untrained average male	3600
Athletically trained average male	4000
Male marathon runner	5100

Figure 84-6 shows the relation between *oxygen consumption* and *total pulmonary ventilation* at different levels of exercise. It is clear from this figure, as would be expected, that there is a linear relation. Both oxygen consumption and total pulmonary ventilation increase about 20-fold between the resting state and maximal intensity of exercise *in the well-trained athlete.*

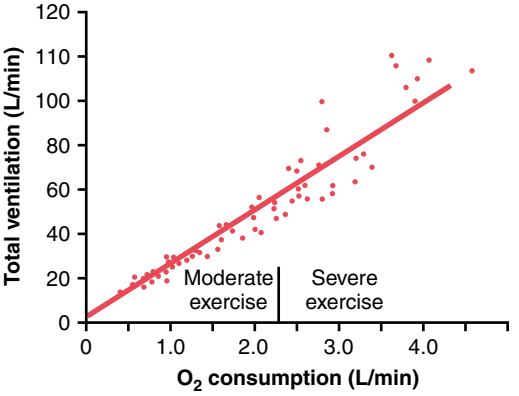

Figure 84-6 Effect of exercise on oxygen consumption and ventilatory rate. (Redrawn from Gray JS: Pulmonary Ventilation and Its Physiological Regulation. Springfield, Ill: Charles C Thomas, 1950.)

Limits of Pulmonary Ventilation. How severely do we stress our respiratory systems during exercise? This can be answered by the following comparison for a normal young man:

	L/min
Pulmonary ventilation at maximal exercise	100-110
Maximal breathing capacity	150-170

Thus, the maximal breathing capacity is about 50 percent greater than the actual pulmonary ventilation during maximal exercise. This provides an element of safety for athletes, giving them extra ventilation that can be called on in such conditions as (1) exercise at high altitudes, (2) exercise under very hot conditions, and (3) abnormalities in the respiratory system.

The important point is that the respiratory system is not normally the most limiting factor in the delivery of oxygen to the muscles during maximal muscle aerobic metabolism. We shall see shortly that the ability of the heart to pump blood to the muscles is usually a greater limiting factor.

Effect of Training on $\dot{V}o_2$ Max. The abbreviation for the rate of oxygen usage under maximal aerobic metabolism is $\dot{V}o_2$ Max. Figure 84-7 shows the progressive effect of athletic training on $\dot{V}o_2$ Max recorded in a group of subjects beginning at the level of no training and then pursuing the training program for 7 to 13 weeks. In this study, it is surprising that the $\dot{V}o_2$ Max increased only about 10 percent. Furthermore, the frequency of training, whether two times or five times per week, had little effect on the increase in $\dot{V}o_2$ Max. Yet, as pointed out earlier, the $\dot{V}o_2$ Max of a marathoner is about 45 percent greater than that of an untrained person. Part of this greater $\dot{V}o_2$ Max of the marathoner probably is genetically determined; that is, those people who have greater chest sizes in relation to body size and stronger respiratory muscles select themselves to become marathoners. However, it is also likely that many years of training increase the marathoner's $\dot{V}o_2$ Max by values considerably greater than the 10 percent that has been recorded in short-term experiments such as that in Figure 84-7.

Oxygen-Diffusing Capacity of Athletes. The *oxygen-diffusing capacity* is a measure of the rate at which oxygen can diffuse from the pulmonary alveoli into the blood. This is expressed in terms of *milliliters of oxygen that will diffuse each minute for each millimeter of mercury difference between alveolar partial pressure of oxygen and pulmonary blood oxygen pressure.* That is, if the partial pressure of oxygen in the alveoli is 91 mm Hg and the oxygen pressure in the blood is 90 mm Hg, the amount of oxygen that diffuses through the respiratory membrane each minute is equal to the diffusing capacity. The following are measured values for different diffusing capacities:

	ml/min
Nonathlete at rest	23
Nonathlete during maximal exercise	48
Speed skaters during maximal exercise	64
Swimmers during maximal exercise	71
Oarsman during maximal exercise	80

The most startling fact about these results is the severalfold increase in diffusing capacity between the resting state and the state of maximal exercise. This results mainly from the fact that blood flow through many of the pulmonary capillaries is sluggish or even dormant in the resting state, whereas in maximal exercise, increased blood flow through the lungs causes all the pulmonary capillaries to be perfused at their maximal rates, thus providing a far greater surface area through which oxygen can diffuse into the pulmonary capillary blood.

It is also clear from these values that those athletes who require greater amounts of oxygen per minute have higher diffusing capacities. Is this because people with naturally greater diffusing capacities choose these types of sports, or is it because something about the training procedures increases the diffusing capacity? The answer is not known, but it is very likely that training, particularly endurance training, does play an important role.

Blood Gases During Exercise. Because of the great usage of oxygen by the muscles in exercise, one would expect the oxygen pressure of the arterial blood to decrease markedly during strenuous athletics and the carbon dioxide pressure of the venous blood to increase far above normal. However, this normally is not the case. Both of these values remain nearly normal, demonstrating the extreme ability of the respiratory system to provide adequate aeration of the blood even during heavy exercise.

This demonstrates another important point: *The blood gases do not always have to become abnormal for respiration to be stimulated in exercise.* Instead, respiration is stimulated mainly by neurogenic mechanisms during exercise, as discussed in Chapter 41. Part of this stimulation results from direct stimulation of the respiratory center by the same nervous signals that are transmitted from the brain to the muscles to cause the exercise. An additional part is believed to result from sensory signals transmitted into the respiratory center from the contracting muscles and moving joints. All this extra nervous stimulation of respiration is normally sufficient to provide almost exactly the necessary increase in pulmonary ventilation required to keep the blood respiratory gases—the oxygen and the carbon dioxide—very near to normal.

Effect of Smoking on Pulmonary Ventilation in Exercise. It is widely known that smoking can decrease an athlete's "wind." This is true for many reasons. First, one effect of nicotine is constriction of the terminal bronchioles of the lungs, which increases the resistance of airflow into and out of the lungs. Second, the irritating effects of the smoke itself

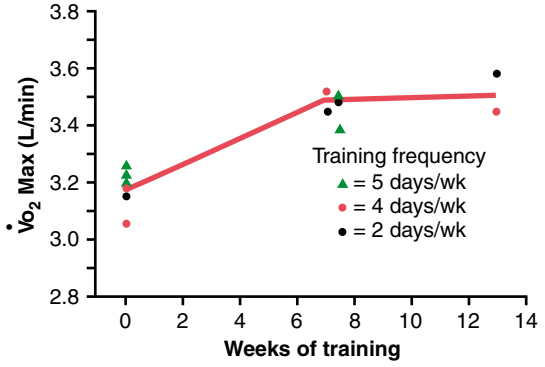

Figure 84-7 Increase in $\dot{V}o_2$ Max over a period of 7 to 13 weeks of athletic training. (Redrawn from Fox EL: Sports Physiology. Philadelphia: Saunders College Publishing, 1979.)

cause increased fluid secretion into the bronchial tree, as well as some swelling of the epithelial linings. Third, nicotine paralyzes the cilia on the surfaces of the respiratory epithelial cells that normally beat continuously to remove excess fluids and foreign particles from the respiratory passageways. As a result, much debris accumulates in the passageways and adds further to the difficulty of breathing. Putting all these factors together, even a light smoker often feels respiratory strain during maximal exercise, and the level of performance may be reduced.

Much more severe are the effects of chronic smoking. There are few chronic smokers in whom some degree of emphysema does not develop. In this disease, the following occur: (1) chronic bronchitis, (2) obstruction of many of the terminal bronchioles, and (3) destruction of many alveolar walls. In severe emphysema, as much as four fifths of the respiratory membrane can be destroyed; then even the slightest exercise can cause respiratory distress. In fact, many such patients cannot even perform the simple feat of walking across the floor of a single room without gasping for breath.

Cardiovascular System in Exercise

Muscle Blood Flow. A key requirement of cardiovascular function in exercise is to deliver the required oxygen and other nutrients to the exercising muscles. For this purpose, the muscle blood flow increases drastically during exercise. Figure 84-8 shows a recording of muscle blood flow in the calf of a person for a period of 6 minutes during moderately strong intermittent contractions. Note not only the great increase in flow—about 13-fold—but also the flow decrease during each muscle contraction. Two points can be made from this study: (1) The actual contractile process itself temporarily decreases muscle blood flow because the contracting skeletal muscle compresses the intramuscular blood vessels; therefore, strong *tonic* muscle contractions can cause rapid muscle fatigue because of lack of delivery of enough oxygen and other nutrients during the continuous contraction. (2) The blood flow to muscles during exercise increases markedly. The following comparison shows the maximal increase in blood flow that can occur in a well-trained athlete.

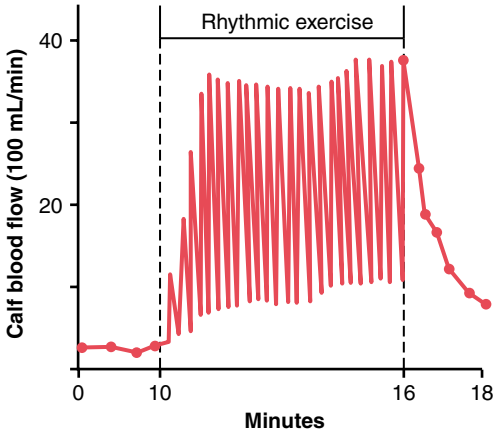

Figure 84-8 Effects of muscle exercise on blood flow in the calf of a leg during strong rhythmical contraction. The blood flow was much less during contraction than between contractions. (Redrawn from Barcroft H, Dornhors AC: Blood flow through human calf during rhythmic exercise. J Physiol 109:402, 1949.)

	ml/100 g Muscle/min
Resting blood flow	3.6
Blood flow during maximal exercise	90

Thus, muscle blood flow can increase a maximum of about 25-fold during the most strenuous exercise. Almost one-half this increase in flow results from intramuscular vasodilation caused by the direct effects of increased muscle metabolism, as explained in Chapter 21. The remaining increase results from multiple factors, the most important of which is probably the moderate increase in arterial blood pressure that occurs in exercise, usually about a 30 percent increase. The increase in pressure not only forces more blood through the blood vessels but also stretches the walls of the arterioles and further reduces the vascular resistance. Therefore, a 30 percent increase in blood pressure can often more than double the blood flow; this multiplies the great increase in flow already caused by the metabolic vasodilation at least another twofold.

Work Output, Oxygen Consumption, and Cardiac Output During Exercise. Figure 84-9 shows the interrelations among work output, oxygen consumption, and cardiac output during exercise. It is not surprising that all these are directly related to one another, as shown by the linear functions, because the muscle work output increases oxygen consumption, and increased oxygen consumption in turn dilates the muscle blood vessels, thus increasing venous return and cardiac output. Typical cardiac outputs at several levels of exercise are the following:

	L/min
Cardiac output in young man at rest	5.5
Maximal cardiac output during exercise in young untrained man	23
Maximal cardiac output during exercise in average male marathoner	30

Thus, the normal untrained person can increase cardiac output a little over fourfold, and the well-trained athlete can increase output about sixfold. (Individual marathoners have been clocked at cardiac outputs as great as 35 to 40 L/min, seven to eight times normal resting output.)

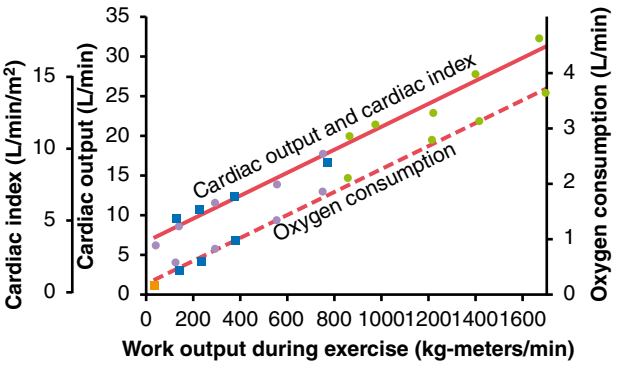

Figure 84-9 Relation between cardiac output and work output (*solid line*) and between oxygen consumption and work output (*dashed line*) during different levels of exercise. (Redrawn from Guyton AC, Jones CE, Coleman TB: Circulatory Physiology: Cardiac Output and Its Regulation. Philadelphia: WB Saunders, 1973.)

Effect of Training on Heart Hypertrophy and on Cardiac Output. From the foregoing data, it is clear that marathoners can achieve maximal cardiac outputs about 40 percent greater than those achieved by untrained persons. This results mainly from the fact that the heart chambers of marathoners enlarge about 40 percent; along with this enlargement of the chambers, the heart mass also increases 40 percent or more. Therefore, not only do the skeletal muscles hypertrophy during athletic training, but so does the heart. However, heart enlargement and increased pumping capacity occur almost entirely in the endurance types, not in the sprint types, of athletic training.

Even though the heart of the marathoner is considerably larger than that of the normal person, resting cardiac output is almost exactly the same as that in the normal person. However, this normal cardiac output is achieved by a large stroke volume at a reduced heart rate. Table 84-2 compares stroke volume and heart rate in the untrained person and the marathoner.

Thus, the heart-pumping effectiveness of each heartbeat is 40 to 50 percent greater in the highly trained athlete than in the untrained person, but there is a corresponding decrease in heart rate at rest.

Role of Stroke Volume and Heart Rate in Increasing the Cardiac Output. Figure 84-10 shows the approximate changes in stroke volume and heart rate as the cardiac output increases from its resting level of about 5.5 L/min to 30 L/min in the marathon runner. The *stroke volume* increases from 105 to 162 milliliters, an increase of about 50 percent, whereas the heart rate increases from 50 to 185 beats/min, an increase of 270 percent. Therefore, the heart rate increase accounts by far for a greater proportion of the increase in cardiac output than does the increase in stroke volume during strenuous exercise. The stroke volume normally reaches its maximum by the time the cardiac output has increased only halfway to its maximum. Any further increase in cardiac output must occur by increasing the heart rate.

Table 84-2 Comparison of Cardiac Function Between Marathoner and Nonathlete

	Stroke Volume (ml)	Heart Rate (beats/min)
Resting		
Nonathlete	75	75
Marathoner	105	50
Maximum		
Nonathlete	110	195
Marathoner	162	185

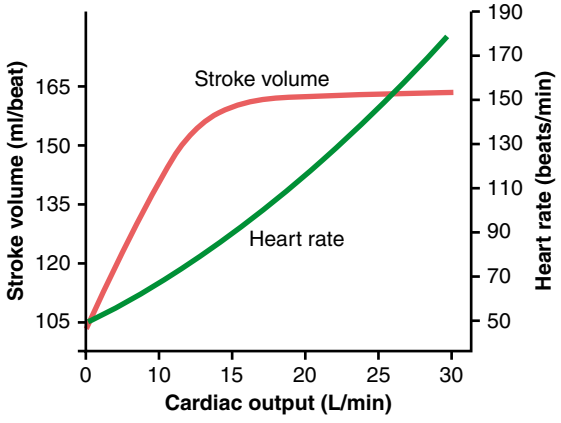

Figure 84-10 Approximate stroke volume output and heart rate at different levels of cardiac output in a marathon athlete.

Relation of Cardiovascular Performance to $\dot{V}O_2$ Max. During maximal exercise, both the heart rate and stroke volume are increased to about 95 percent of their maximal levels. Because the cardiac output is equal to stroke volume *times* heart rate, one finds that the cardiac output is about 90 percent of the maximum that the person can achieve. This is in contrast to about 65 percent of maximum for pulmonary ventilation. Therefore, one can readily see that the cardiovascular system is normally much more limiting on $\dot{V}O_2$ Max than is the respiratory system, because oxygen utilization by the body can never be more than the rate at which the cardiovascular system can transport oxygen to the tissues.

For this reason, it is frequently stated that the level of athletic performance that can be achieved by the marathoner mainly depends on the performance capability of his or her heart, because this is the most limiting link in the delivery of adequate oxygen to the exercising muscles. Therefore, the 40 percent greater cardiac output that the marathoner can achieve over the average untrained male is probably the single most important physiologic benefit of the marathoner's training program.

Effect of Heart Disease and Old Age on Athletic Performance. Because of the critical limitation that the cardiovascular system places on maximal performance in endurance athletics, one can readily understand that any type of heart disease that reduces maximal cardiac output will cause an almost corresponding decrease in achievable total body muscle power. Therefore, a person with congestive heart failure frequently has difficulty achieving even the muscle power required to climb out of bed, much less to walk across the floor.

The maximal cardiac output of older people also decreases considerably—there is as much as a 50 percent decrease between ages 18 and 80. Also, there is even more decrease in maximal breathing capacity. For these reasons, as well as reduced skeletal muscle mass, the maximal achievable muscle power is greatly reduced in old age.

Body Heat in Exercise

Almost all the energy released by the body's metabolism of nutrients is eventually converted into body heat. This applies even to the energy that causes muscle contraction for the following reasons: First, the maximal efficiency for conversion of nutrient energy into muscle work, even under the best of conditions, is only 20 to 25 percent; the remainder of the nutrient energy is converted into heat during the course of the intracellular chemical reactions. Second, almost all the energy that does go into creating muscle work still becomes body heat because all but a small portion of this energy is used for (1) overcoming viscous resistance to the movement of the muscles and joints, (2) overcoming the friction of the blood flowing through the blood vessels, and (3) other, similar effects—all of which convert the muscle contractile energy into heat.

Now, recognizing that the oxygen consumption by the body can increase as much as 20-fold in the well-trained athlete and that the amount of heat liberated in the body is almost exactly proportional to the oxygen consumption (as discussed in Chapter 72), one quickly realizes that tremendous amounts of heat are injected into the internal body tissues when performing endurance athletic events. Next, with a vast rate of heat flow into the body, on a very hot and humid day so that the sweating mechanism cannot eliminate the heat, an intolerable and even lethal condition called *heatstroke* can easily develop in the athlete.

Heatstroke. During endurance athletics, even under normal environmental conditions, the body temperature often rises from its normal level of 98.6° to 102° or 103°F (37° to 40°C). With very hot and humid conditions or excess clothing, the body temperature can easily rise to 106° to 108°F (41° to 42°C). At this level, the elevated temperature itself becomes destructive to tissue cells, especially the brain cells. When this happens, multiple symptoms begin to appear, including extreme weakness, exhaustion, headache, dizziness, nausea, profuse sweating, confusion, staggering gait, collapse, and unconsciousness.

This whole complex is called *heatstroke,* and failure to treat it immediately can lead to death. In fact, even though the person has stopped exercising, the temperature does not easily decrease by itself. One of the reasons for this is that at these high temperatures, the temperature-regulating mechanism itself often fails (see Chapter 73). A second reason is that in heatstroke, the very high body temperature approximately doubles the rates of all intracellular chemical reactions, thus liberating still more heat.

The treatment of heatstroke is to reduce the body temperature as rapidly as possible. The most practical way to do this is to remove all clothing, maintain a spray of cool water on all surfaces of the body or continually sponge the body, and blow air over the body with a fan. Experiments have shown that this treatment can reduce the temperature either as rapidly or almost as rapidly as any other procedure, although some physicians prefer total immersion of the body in water containing a mush of crushed ice if available.

Body Fluids and Salt in Exercise

As much as a 5- to 10-pound weight loss has been recorded in athletes in a period of 1 hour during endurance athletic events under hot and humid conditions. Essentially all this weight loss results from loss of sweat. Loss of enough sweat to decrease body weight only 3 percent can significantly diminish a person's performance, and a 5 to 10 percent rapid decrease in weight can often be serious, leading to muscle cramps, nausea, and other effects. Therefore, it is essential to replace fluid as it is lost.

Replacement of Sodium Chloride and Potassium. Sweat contains a large amount of sodium chloride, for which reason it has long been stated that all athletes should take salt (sodium chloride) tablets when performing exercise on hot and humid days. However, overuse of salt tablets has often done as much harm as good. Furthermore, if an athlete becomes acclimatized to the heat by progressive increase in athletic exposure over a period of 1 to 2 weeks rather than performing maximal athletic feats on the first day, the sweat glands also become acclimatized, so the amount of salt lost in the sweat becomes only a small fraction of that lost before acclimatization. This sweat gland acclimatization results mainly from increased aldosterone secretion by the adrenal cortex. The aldosterone in turn has a direct effect on the sweat glands, increasing reabsorption of sodium chloride from the sweat before the sweat itself issues forth from the sweat gland tubules onto the surface of the skin. Once the athlete is acclimatized, only rarely do salt supplements need to be considered during athletic events.

Experience by military units exposed to heavy exercise in the desert has demonstrated still another electrolyte problem—the loss of potassium. Potassium loss results partly from the increased secretion of aldosterone during heat acclimatization, which increases the loss of potassium in the urine, as well as in the sweat. As a consequence of these findings, some of the supplemental fluids for athletics contain properly proportioned amounts of potassium along with sodium, usually in the form of fruit juices.

Drugs and Athletes

Without belaboring this issue, let us list some of the effects of drugs in athletics.

First, *caffeine* is believed by some to increase athletic performance. In one experiment on a marathon runner, running time for the marathon was improved by 7 percent by judicious use of caffeine in amounts similar to those found in one to three cups of coffee. Yet experiments by others have failed to confirm any advantage, thus leaving this issue in doubt.

Second, use of *male sex hormones (androgens)* or other anabolic steroids to increase muscle strength undoubtedly can increase athletic performance under some conditions, especially in women and even in men. However, anabolic steroids also greatly increase the risk of cardiovascular damage because they often cause hypertension, decreased high-density blood lipoproteins, and increased low-density lipoproteins, all of which promote heart attacks and strokes.

In men, any type of male sex hormone preparation also leads to decreased testicular function, including both decreased formation of sperm and decreased secretion of the person's own natural testosterone, with residual effects sometimes lasting at least for many months and perhaps indefinitely. In a woman, even more dire effects can occur because she is not normally adapted to the male sex hormone—hair on the face, a bass voice, ruddy skin, and cessation of menses.

Other drugs, such as *amphetamines* and *cocaine,* have been reputed to increase one's athletic performance. It is equally true that overuse of these drugs can lead to deterioration of performance. Furthermore, experiments have failed to prove the value of these drugs except as a psychic stimulant. Some athletes have been known to die during athletic events because of interaction between such drugs and the norepinephrine and epinephrine released by the sympathetic nervous system during exercise. One of the possible causes of death under these conditions is overexcitability of the heart, leading to ventricular fibrillation, which is lethal within seconds.

Body Fitness Prolongs Life

Multiple studies have now shown that people who maintain appropriate body fitness, using judicious regimens of exercise and weight control, have the additional benefit of prolonged life. Especially between the ages of 50 and 70, studies have shown mortality to be three times less in the most fit people than in the least fit.

But why does body fitness prolong life? The following are some of the most important reasons.

Body fitness and weight control greatly reduce cardiovascular disease. This results from (1) maintenance of moderately lower blood pressure and (2) reduced blood cholesterol and low-density lipoprotein along with increased high-density lipoprotein. As pointed out earlier, these changes all work together to reduce the number of heart attacks, brain strokes, and kidney disease.

The athletically fit person has more bodily reserves to call on when he or she does become sick. For instance, an 80-year-old nonfit person may have a respiratory system that limits oxygen delivery to the tissues to no more than 1 L/min; this means a *respiratory reserve of no more than threefold to fourfold.* However, an athletically fit old person may have twice as much reserve. This is especially important in preserving life when the older person develops conditions such as pneumonia that can rapidly require all available respiratory reserve. In addition, the ability to increase cardiac output in times of need (the "cardiac reserve") is often 50 percent greater in the athletically fit old person than in the nonfit person.

Exercise and overall body fitness also reduce the risk for several chronic metabolic disorders associated with obesity such as insulin resistance and type II diabetes. Moderate exercise, even in the absence of significant weight loss, has been shown to improve insulin sensitivity and reduce, or in some cases eliminate, the need for insulin treatment in patients with type II diabetes.

Improved body fitness also reduces the risk for several types of cancers, including breast, prostate, and colon cancer. Much of the beneficial effects of exercise may be related to reduction in obesity. However, studies in experimental animals and in humans have also shown that regular exercise reduces the risk for many chronic diseases through mechanisms that are incompletely understood but are, at least to some extent, independent of weight loss or decreased adiposity.

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